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Original article

# Effects of acute aerobic exercise on motor response inhibition: An ERP study using the stop-signal task

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**Abstract**

*Purpose*: The purpose of this study was to determine the effects of acute exercise on motor response inhibition using both behavioral and electrophysiological approaches.
*Methods*: The P3 and N1 event-related potential (ERP) components were recorded while performing a stop-signal task in 21 college students following a moderately intense acute exercise bout for 30 min and a sedentary control session that involved reading.
*Results*: Acute exercise induced a shorter stop signal response time (SSRT) as compared to control; however, the go response time (Go RT) remained unchanged. In examining the ERP data, acute exercise increased both P3 amplitude and latency but did not affect the N1 component.
*Conclusion*: Acute exercise has a selective and beneficial effect on cognitive function, specifically affecting the motor response inhibition aspect of executive function. Furthermore, acute exercise predominately impacts later stages of information processing during motor response inhibition, which may lead to an increase in attentional resource allocation and confer the ability to successfully withhold a response to achieve motor response inhibition.


*Keywords:* Cognition; Executive function; Inhibitory control; N1; P3

## 1. Introduction

Previous studies have suggested a beneficial effect on cognition following acute exercise.[1,2] This observation is supported by meta-analytic reviews in which acute exercise yielded a significant and positive impact on cognitive function, with a mean overall effect size ranging from 0.1 to 0.2.[3,4] Despite the well-established relationship between acute exercise and cognition, previous studies have focused on basic cognitive functions. Recently, however, some groups have shifted their attention to higher cognitive processes, such as executive function.[5,6]

Executive function is an essential cognitive process that involves the control and regulation of other more basic cognitive processes. It is responsible for the ability to respond appropriately to situations in which conflicts arise and embraces a large variety of multi-faceted constructs.[7,8] While several distinct sub-components of executive function have been proposed,[9] inhibition has been consistently recognized as a primary aspect of executive function. Inhibition requires both the ability to resolve conflicting responses from unrelated or distracting stimuli and the ability to suppress improper behaviors.[8,10,11] Dysfunctional inhibition has been linked to several psychiatric disorders, including schizophrenia[12] and attention-deficit/hyperactivity disorder.[13]

Recent studies have suggested that acute exercise could potentially impact one's inhibitory ability as measured by various cognitive tasks, such as the Stroop Task,[14–18] the Eriksen Flanker Task,[19–21] the go/no-go task[22–24] and the

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stop-signal task.[25] Yet, it is worthy to note that these tasks, which all measure inhibition, may actually measure different particular inhibitory processes.[26] For example, the Stroop and Eriksen Flanker tasks are thought to assess the ability to resolve conflicting stimuli and therefore measure a more cognitive form of inhibition called interference inhibition. In contrast, the go/no-go and stop-signal tasks are thought to measure motor response inhibition.[11] Based upon the notion that acute exercise might selectively impact specific aspects of executive function as proposed by Etnier and Chang,[5] acute exercise might affect only a specific inhibitory process or function. If so, the effects would only be observed when using a select task that is sensitive to those effects. Given that Stroop, Eriksen Flanker, and go/no-go tasks have been primarily utilized in studying acute exercise and inhibition, the aim of this study was to examine motor response inhibition using the stop-signal task in order to advance the knowledge base.

The stop-signal task has been widely used to measure motor response inhibition[27,28] by employing computational modeling of a dynamic tracking algorithm which takes into account participants' strategic adjustments during the task. In contrast to the go/no-go task, which presents either "go" or "no-go" signals and requires participants to stop executing one set and respond to a different set of stimuli, the stop-signal task presents "go" signals in both "go" and "stop" trials and requires participants to terminate an already executed response when a "stop" signal unpredictably appears after a delay in "stop" trials. Therefore, the go/no-go task measures overall motor response inhibition[11] and provides an index of inhibition failure,[29] whereas the stop-signal task increases the difficulty of inhibiting the "go" response and measures a later stage of inhibition.[30] To date, only one study has utilized the stop-signal task to examine the effects of acute exercise on response inhibition.[25] In that study, Joyce et al.[25] observed that 30 min of acute moderate-intensity exercise failed to affect basic information processes (i.e., go response time (Go RT)) but improved inhibitory performance (i.e., shorter stop signal response time (SSRT)), suggesting that acute exercise has a selectively beneficial effect on overall motor response inhibition.

Due to their precise temporal resolution, measuring event-related potentials (ERPs) could reveal the underlying mechanisms by which acute exercise affects the brain and cognitive performance. ERPs allow researchers to study patterns of neuronal activity by measuring voltage changes in response to or in preparation for an event (i.e., a stimulus). Previous studies examining inhibition have suggested that acute exercise is associated with increased neuronal activity.[19−21] For example, acute exercise has been shown to increase the amplitude of the largest positive component in the human ERP waveform (i.e., the P3 component), which occurs approximately 300−800 ms after stimulus presentation. Because the amplitude of the P3 component is believed to reflect the allocation of attentional resources during stimulus engagement,[31−33] acute exercise induces a greater allocation of attentional resources to the given task.[19−21,34]

Most prior studies have focused on P3 and only a few studies have examined other ERP components, such as the N1. Unlike P3, which is a late and endogenous ERP component, N1 is an exogenous component that is associated with an earlier cognitive processing stage and reflects the initial extraction of sensory information.[35] Previous exercise studies have revealed that acute exercise has a limited influence on the N1 elicited by a choice reaction time task; however, no acute exercise study to date has investigated inhibitory processes such as are required in the stop-signal task using this ERP component.[36,37] This is important because the N1 component is thought to represent the amount of spatial attention that is directed toward a visual stimulus (e.g., stop signal), such that a larger amplitude of N1 would translate to more attention shifted to the stop signal.[38] This interpretation of the N1 component suggests that the attention oriented toward the stop signal could profoundly influence the engagement or success of inhibitory processes during the stop-signal task. Taken together, the effect of acute exercise on inhibition in the context of the N1 component elicited by the stop-signal task are still unknown, yet this information about the contribution of early information processing in motor response inhibition is critical to our understanding of acute exercise effects on inhibition and executive function.

The purpose of this study was to clarify the effects of acute exercise on motor inhibition. Specifically, a stop-signal task, requiring a general motor response inhibition, was performed after a single bout of moderately intense aerobic exercise. The precise temporal resolution of motor inhibitory processes was captured by ERPs recorded during the stop-signal task. Our hypothesis was that acute exercise would not only facilitate overall motor inhibition, but would do so by positively influencing specific ERP components that reflect both late (P3) and early (N1) information processing stages.

## 2. Methods

### 2.1. Participants

Twenty-one right-handed college students (19−24 years old) were recruited through flyers from regions surrounding Taoyuan, New Taipei City, and Taipei. All participants provided written informed consent prior to their involvement in this study in accordance with the Institutional Review Board of National Taiwan University. All participants completed the Physical Activity Readiness Questionnaire (PAR-Q) and the Health Screening Questionnaire (HSQ) to determine if they had any conditions that would prevent them from participating in acute exercise or the cardiovascular fitness assessment. Only those participants who met the "low risk" criteria set by the American College of Sports Medicine guidelines were eligible to participate in this study.[39] Additionally, all participants had normal or corrected-to-normal vision (i.e., 20/20), were non-smokers, were not currently taking any medication, and had no history of substance abuse or mental health disorders.

Demographic data that might influence cognitive function were collected, and both intelligence and physical activity levels were assessed by the Digit Span Forward and Backward tests from the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III)[40] along with the International Physical Activity Questionnaire.[41] Demographic, intelligence, and fitness data are presented in Table 1.

### 2.2. Cardiovascular fitness assessment

Participants completed a single-stage submaximal treadmill walking test (SSTWT). The SSTWT, developed by Ebbeling et al.,[42] is commonly used to predict maximal oxygen consumption ($VO_{2peak}$) because estimated $VO_{2peak}$ values from this test have been highly correlated with directly measured $VO_{2peak}$ ($r = 0.96$, with a multiple correlation 0.86).[43]

The SSTWT protocol consists of two 4-min stages. During the first stage, participants were instructed to warm up on the treadmill at a self-selected walking speed of between 2.0 and 4.5 mph at a 0% grade. The speed of the treadmill was increased until heart rate (HR) was raised to 60%−70% of participants' age-predicted maximum heart rate ($HR_{max}$), which was estimated based on the formula (207− (0.67 × age)).[44] Throughout the second 4-min stage, participants were required to maintain the speed they had established during the first stage while the treadmill was raised to a 5% grade. Steady-state heart rate (SSHR) was measured during the last 2 min of the second stage, and together with participant age, gender, and final treadmill speed, was used to predict $VO_{2peak}$ using the following formula:

$VO_{2peak}$ (mL/kg/min) = 15.1 + 21.8 × Speed (mph) − 0.327 × SSHR (bpm) − 0.263 × Speed × Age (year) + 0.00504 × SSHR × Age + 5.98 × Gender (0 = female; 1 = male).

### 2.3. Stop-signal task

Our modified stop-signal task was adapted from Johnstone et al.[45] (Fig. 1). Participants were instructed to focus on a central fixation cross on the screen and avoid body movements during electroencephalographic (EEG) recording. The stop-signal task consisted of two blocks of 200 trials (total of 400 trials) that lasted approximately 25 min total. Each trial began with the presentation of a central fixation for 500 ms followed by stimuli (i.e., "go" signal of "go" trial, "go" and "stop" signals of "stop" trial) with 196 × 42 pixels (visual angle of 3.65) for 500 ms and a white screen for 1400 ms.

Table 1
Demographic and fitness characteristics of study participants (means ± SD).

| Variables | Male ($n = 19$) | Female ($n = 2$) | Total ($n = 21$) |
|---|---|---|---|
| Age (year) | 21.67 ± 4.92 | 20.00 ± 0.00 | 21.50 ± 4.68 |
| Height (cm) | 171.11 ± 5.07 | 160.00 ± 5.66 | 170.00 ± 6.03 |
| Weight (kg) | 68.00 ± 9.41 | 53.00 ± 7.07 | 66.50 ± 10.15 |
| BMI (kg/m$^2$) | 23.20 ± 2.88 | 20.64 ± 1.30 | 22.94 ± 2.85 |
| Digit span (forward) | 14.00 ± 1.58 | 16.00 ± 0.00 | 14.21 ± 1.62 |
| Digit span (backward) | 9.11 ± 3.80 | 12.50 ± 2.12 | 9.45 ± 3.77 |
| $VO_{2peak}$ (mL/kg/min) | 56.03 ± 9.82 | 46.37 ± 1.04 | 55.01 ± 9.75 |

Two types of trials, "go" and "stop", were randomly presented one at a time against a white background in the center of a 17-inch computer monitor placed 70 cm in front of participants at eye-level. To increase the potency of the conflicting stimulus, approximately 25% (i.e., 50 trials) were "stop" trials and 75% (i.e., 150 trials) were "go" trials. During a "go" trial, a left- or right-pointing black arrow (i.e., "go" signal) appeared in the center of the computer screen with equal probability, and participants were required to press the right or left key based on the direction of the arrow. When a "stop" trial occurred, a red square appeared shortly after the appearance of the arrow ("stop" signal), and participants were instructed to inhibit their response to the primary "go" signal, regardless of which direction the arrow was pointing. The presentation of the "go" and "stop" trials was randomized (with 0.75 and 0.25 probability, respectively) to avoid establishing expectations.

The time interval between the appearance of the arrow and the appearance of the red square (also known as the stop-signal delay or SSD) varied based on a dynamic tracking algorithm which either decreased or increased the SSD by 50 ms (note: shorter SSDs are easier than longer SSDs because they appear more closely in time to the presentation of the arrow). The initial SSD was set at 200 ms at the start of each experimental block, and the duration of the next SSD (ranging from 50 to 450 ms) depended upon whether the participant failed (−50 ms) or succeeded (+50 ms) in inhibiting his/her response in the preceding "stop" trial. The dynamic tracking algorithm ensured that approximately 50% of the "stop" trials resulted in successful inhibition. Participants were instructed to respond as quickly and accurately as possible to the "go" signal or to inhibit their response when a red square appeared (i.e., the "stop" signal).

The Go RT of "go" trial and SSRT were the main indices of the stop-signal task. The SSRT, the index of inhibitory ability, was estimated using the following equation: SSRT = mean Go RT − mean SSD, while the Go RT of "go" trials was determined by measuring the time interval between when the "go" signal appeared and when participants provided the correct response. Errors of commission and omission were excluded from the analyses.

### 2.4. ERP data acquisition

EEG recordings were conducted in a quiet and sound-attenuated room using an elastic cap (Quick-Cap; NeuroScan Inc., El Paso, TX, USA) affixed to 32 Ag/AgCl electrodes that were designed to conform with the international 10-20 system.[46] Continuous EEG measurements were taken, averaged from the right and left mastoids, and the ground electrode was located on the mid-forehead. Eye movements were monitored by two pairs of electrodes. The vertical electrooculogram (VEOG), used to detect eye blinks, was recorded from electrodes that were attached in a straight line below and above the left eye, whereas the horizontal electrooculogram (HEOG) was monitored through two electrodes placed in a straight line at the outer canthi of both eyes. The impedance for all

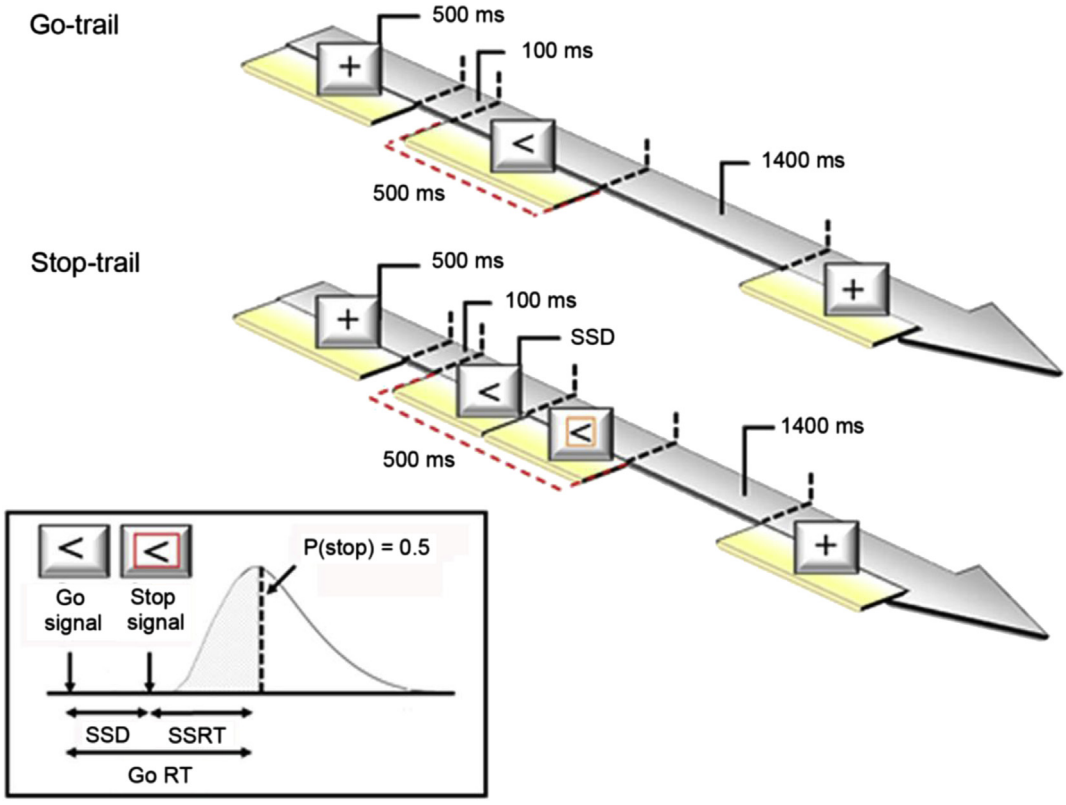


Fig. 1. Schematic of the stimulus and timeline of the stop-signal task. The curve represents the distribution of the speed of the Go process. P(stop) = probability of successful inhibition; SSD = stop-signal delay; SSRT = stop-signal response time; Go RT = go response time.

electrodes was kept under 10 kΩ throughout the recording period, and the resulting continuous signals were amplified by a SynAmps EEG amplifier using the Scan 4.5 package (NeuroScan Inc.). The digitization was set at a 500 Hz sampling rate, and a 60-Hz notch filter was applied to remove additional electrical noise.

The EEG data were segmented offline into epochs for time-locked ERP component by defining as 100 ms pre- to 900 post- "stop" signal onset in which the analysis of time points of the stop-signal was set at average of SSD in strop trail. The data were then passed through a band-pass filter from 1 to 20 Hz (24 dB/Oct), and a baseline correction was performed with the 100 ms pre-stimulus time period. Any trials in which the amplitude exceeded ±100 μV and any trials with incorrect responses were excluded from analyses.

The key ERP components of interest for correct "stop" trials in this study included the N1 and P3 components. The amplitude of these components were quantified within a predetermined latency window using an automatic peak-defining program across the Fz, Cz, and Pz sites.[20,21] Specifically, N1 was defined as the largest negative voltage in the 50−150 ms latency window from "stop" signal onset, whereas P3 was defined as the largest positive voltage in the 250−550 ms latency window from "stop" signal onset. Additional topographic distributions of the grand average were drawn from the global scalp data with all 32 electrodes.

### 2.5. Experimental procedure

Each participant individually visited our laboratory at the National Taiwan Sport University on three separate occasions that occurred at least 24 h apart. During the first visit, the experimenter gave a brief introduction to the study and participants completed a statement of informed consent and filled out demographic, PAR-Q, and HSQ questionnaires. All participants who met the initial inclusion criteria then performed two practice blocks of 24 trials each to familiarize themselves with the cognitive task. Next, a Polar HR monitor (Sport Tester PE 3000; Polar Electro Oy, Kempele, Finland) was affixed and participants' resting HR during the last minute of 10 min of quietly sitting in a comfortable chair was established. Lastly, participants underwent an SSTWT to determine peak cardiovascular fitness ($VO_{2peak}$).

The exercise session and the control session were randomly assigned on the second or third visit to eliminate any bias based on the order (i.e., learning or order effect). These two visits occurred at approximately the same time of day for each participant. During the exercise session, participants engaged in 30 min of aerobic exercise on a motor-driven treadmill in a temperature-controlled room (mean temperature 22 °C ), which consisted of a 5-min warm-up phase, a 20-min main exercise phase, and a 5-min cool-down phase. During the warm-up phase, the treadmill speed was set at 2.5 mph and was increased until the exercise intensity rose to 65%−75% of the

participant's age-predicted $HR_{max}$ and this was maintained during the main exercise phase. Exercise at this target HR has been defined as moderate intensity[39] and has been shown to positively affect cognition.[47–49] Both HR (i.e., in-task HR) and the Borg[50] rating of perceived exertion (RPE) were recorded every 2 min during the exercise session. In contrast, participants in the control session were instructed to read exercise-related articles for 30 min, while in-task HR was recorded.

Within 10 min following exercise and reading sessions, participants were comfortably seated in a chair with supports for their hands and arms in a dimly lit, sound-proof, electrically-shielded room. The cognitive task was then administered and EEG recordings were taken throughout the task performance period. Each participant was compensated approximately US$20, and the goals of the study were briefly explained at the end of the final visit.

### 2.6. Statistical analyses

Descriptive statistics are presented as in Table 1. A 2 (session: exercise, control) × 2 (time point: resting HR, in-task HR) repeated-measures analysis of variance (ANOVA) was performed to compare HR values before and during exercise sessions and as a check of exercise intensity.

Paired sample *t* tests were performed to compare the mean differences between exercise and control sessions for SSRT and Go RT. For the ERP components, 2 (session: exercise, control) × 3 (site: Fz, Cz, Pz) repeated-measures ANOVAs were conducted for correct trials when the stop signal was displayed with session and site as within-subject factors and peak amplitude and latency of the N1 and P3 components as dependent variables. Greenhouse Geisser corrections were performed to correct for non-sphericity. Finally, *post hoc* comparisons with multiple *t* tests were conducted, and the $\alpha$ level was set at 0.05 prior to Bonferroni correction.

## 3. Results

### 3.1. Exercise effects on HR and RPE

The 2 (session) × 2 (time point) ANOVA revealed significant effects for session ($F = 1077.31$, $p < 0.001$), time point ($F = 514.04$, $p < 0.001$), and an interaction between session and time point ($F = 1175.51$, $p < 0.001$). Follow-up analyses revealed that in-task HR measured during the exercise session (155.54 ± 6.05 beats/min) was significantly higher than resting HR (69.31 ± 5.74 beats/min), but no time point difference was observed for the control session (in-task HR: 68.15 ± 4.76 beats/min, resting HR: 68.15 ± 4.76 beats/min). Additionally, the session difference was only observed for in-task HR, with higher HR values observed for the exercise session relative to control. The average RPE in the exercise session was 13.24 ± 1.92.

### 3.2. Behavioral data

A *t* test revealed that participants' SSRT after exercise (219.06 ± 54.57 ms) was significantly shorter than after the control condition (249.01 ± 67.08 ms) ($t = 3.35$, $p < 0.003$). However, the Go RT was not significantly different after exercise and control sessions (485.41 ± 120.23 ms *vs.* 465.53 ± 156.91 ms) ($t = -1.06$, $p > 0.05$, Fig. 2). The averaged SSDs were 266.34 ms and 216.52 ms for exercise and control session, respectively.

### 3.3. ERP data

#### 3.3.1. N1 component

The N1 amplitude of the ERP data was not affected by exercise as determined by a 2 (session) × 3 (site) ANOVA, which revealed no significant effects of session ($F = 0.31$, $p > 0.05$) and the interaction between session and site ($F = 0.71$, $p > 0.05$). Only a main effect of ERP site ($F = 10.96$, $p < 0.001$), with larger amplitude at Fz and Pz (1.72 ± 1.17, 2.97 ± 0.78) than Cz (0.34 ± 0.98) was revealed.

The N1 latency was similarly unaffected as determined by a 2 (session) × 3 (site) ANOVA, which revealed no significant effects of session ($F = 3.27$, $p > 0.05$), ERP site ($F = 0.45$, $p > 0.05$), or the interaction between session and site ($F = 1.34$, $p > 0.05$) (Fig. 3).

#### 3.3.2. P3 component

A 2 (session) × 3 (site) ANOVA revealed a significant effect of session ($F = 13.93$, $p = 0.002$), with the exercise session (12.60 ± 1.68 μV) resulting in a larger P3 amplitude than the control session (7.49 ± 1.31 μV). The ERP site also resulted in a significant effect ($F = 20.44$, $p = 0.001$) with Fz (11.90 ± 1.56 μV) having a largest amplitude, followed by Pz (10.34 ± 1.31 μV), and Cz having a smallest amplitude (7.91 ± 1.31 μV). However, no significant difference was observed for the interaction between session and site ($F = 1.62$, $p > 0.05$).

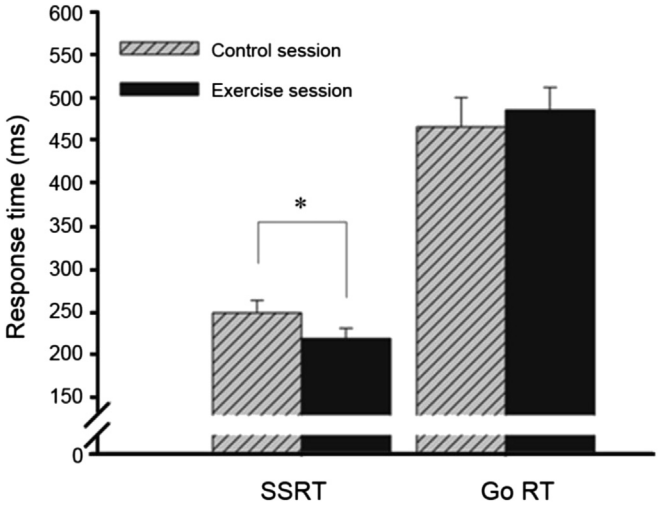


Fig. 2. Comparison of the stop signal response time (SSRT) and go response time (Go RT) between acute exercise and resting control sessions (means ± SE). *$p < 0.003$.

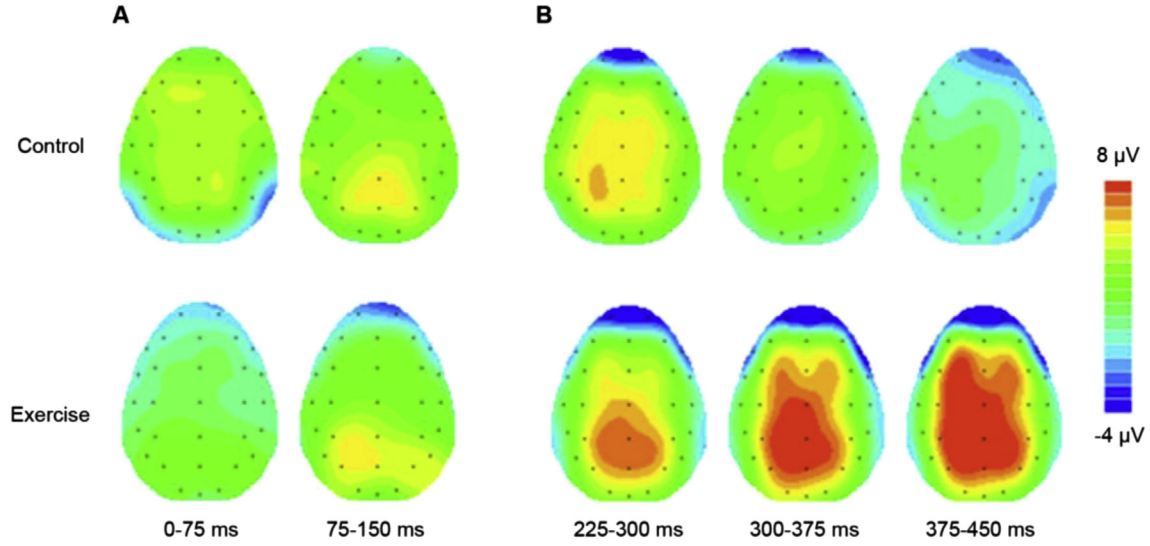


Fig. 3. Topographic distribution of the measured voltage amplitudes following "stop" signal for the control and exercise sessions in (A) N1 (0−150 ms) and (B) P3 (225−450 ms), respectively.

P3 component latency was similarly affected by exercise. A 2 (session) × 3 (site) ANOVA revealed that session once again had a significant effect ($F = 5.09$, $p = 0.03$), with exercise (390.77 ± 20.22 ms) resulting in a longer P3 latency than control (326.65 ± 14.78 ms), but no main effect of ERP site ($F = 2.79$, $p > 0.05$). A significant interaction between session and site ($F = 5.35$, $p = 0.01$) were also revealed. With follow-up *post hoc* test indicated that no session difference was observed in Fz and Cz, but exercise session had longer P3 latency than control in Pz (390.49 ± 25.48 ms, 276.82 ± 10.56 ms). Additionally, no ERP site difference was observed in exercise session, but Fz and Cz (353.15 ± 25.05 ms, 349.98 ± 19.15 ms) have longer P3 latency than Pz (276.82 ± 10.56 ms) in control session.

## 4. Discussion

The main objective of this study was to assess the effects of acute exercise on the inhibitory aspect of executive function using behavioral and electrophysiological approaches. Specifically, this study was the first to use the stop-signal task to examine the underlying neural mechanisms of the effects of moderately intense, acute aerobic exercise on motor response inhibition. The results revealed that acute exercise results in a shorter SSRT but does not alter the Go RT. Acute exercise also increases both P3 amplitude and latency but did not alter the N1 component.

The observation that SSRT is shorter after acute exercise when compared to control is consistent with a previous study demonstrating that 30 min of acute, moderately intense exercise on a cycle ergometer enhanced stop-signal performance and resulted in a shorter SSRT.[25] These behavioral findings suggest that acute exercise enhances the ability to control motor response inhibition.[51] Interestingly, we found no significant difference in Go RT between exercise and control sessions, which also corroborates the observations by Joyce et al.[25] that acute exercise does not affect the Go RT. The horse-race model proposed by Logan and Cowan[52] has been widely used to understand the stop-signal paradigm. This model is based on the idea that the ability to inhibit a response during the stop-signal task is probabilistic and depends upon variability in the response times of the stop and go processes, as well as the SSD. The amount of time that elapses between the appearance of the stop signal and the subject's response is defined as the SSRT, which is calculated from the Go RT and the SSD. In the horse-race model, the "stop" and "go" processes are considered to be independent from each other. While the "stop" process is cognitively demanding and requires the ability to process the stop signal and engage response inhibition, the "go" process is less cognitively demanding, is triggered by the go signal, and involves more basic information processing. The behavioral outcome is determined by the "race" between these two processes. Consequently, if the "stop" process finishes before the "go" process during a stop trial, then the response will be successfully withheld; otherwise, a response will be executed.[53,54] Because we observed shorter SSRTs after exercise and the Go RT was not found to change between exercise and control sessions, we conclude that acute exercise facilitates the "stop" processing instead of altering the "go" processing. In other words, rather than improve cognitive performance in general, acute exercise selectively benefits the motor response inhibition aspect of executive function.

Our ERP data revealed that acute exercise leads to a larger P3 amplitude, which agrees with prior studies that examined inhibition using either the go/no-go task[24] or the flanker task.[19−21,23] Notably, we observed that both the ERP site and the type of treatment had a significant effect on the amplitude. The largest P3 amplitude during the stop-signal task was observed at Fz and the finding of the more frontal P3 component when a response was successfully withheld corroborates findings from previous studies.[55,56] P3 amplitude is

thought to proportionally reflect the amount of attentional resources that are devoted to a given task.[19,21] The P3 component also represents cognitive processes that may be positively associated with the successful inhibition of the stop-signal task.[56,57] Accordingly, our observation of increased P3 amplitude after exercise suggests that acute exercise promotes motor response inhibition by more efficiently allocating attentional resources. Specifically, the increase in attentional resources devoted to inhibition in the stop-signal task likely equates to similar observations in previously studied inhibition-related tasks.

Interestingly, we observed a longer parietal P3 latency following acute exercise when compared to the control session, which contradicts previous studies involving alternate inhibition-related tasks. For example, Kamijo et al.[24] failed to observe an effect of acute exercise on P3 latency in the go/no-go task. In contrast, other studies have reported a shorter P3 latency after acute exercise in the flanker task.[19–21,23] These inconsistent findings among the three tasks might be related to the specific characteristics of inhibition required for each task; in other words, P3 latency may be task-dependent. By varying the timing of the stop signal presentation, Ramautar et al.[55] observed an increase in both the P3 amplitude and latency when the stop signals were presented less frequently. According to the horse-race model, fewer stop signals would result in a stronger bias towards the go stimulus and would in turn require more inhibitory pressure to withhold the propensity to yield a rapid go response. Therefore, to overcome the lower probability of the more difficult condition and the resulting strong response bias, more attentional resource allocation (i.e., larger P3 amplitude) and a longer information processing time (i.e., delayed P3 latency) were needed. Indeed, Ramautar et al.[57] detected a positive correlation between the increased stop-P3 latency and the SSD. Therefore, our observation that P3 latency increased after acute exercise may reflect a successful withholding of the inhibitory response at the neuronal level and a lengthening of the SSD. This withholding would in turn facilitate response inhibition, which would be reflected by the shorter SSRT (i.e., SSRT = mean Go RT−mean SSD).

Novel to the present study is the examination of both N1 and P3 components of the ERP waveform. In contrast to the P3, which was affected by acute exercise, neither N1 amplitude or latency were altered by acute exercise. The N1 component is believed to reflect the initial sensory extraction of a stimulus[58] and is associated with selective attention and discrimination processes during the early stages of visual information processing.[59] Therefore, our lack of significant findings for the N1 component suggests that acute exercise may not impact the perception of attention to a stimulus during early information processing, which is supported by previous studies in which acute exercise failed to affect N1.[36,37] While these early ERP studies involved a visual oddball task that examined relatively basic information processing, our findings suggest that the lack of an acute exercise-dependent effect on N1 can also be extended to the motor response inhibition aspect of executive function.

There are several limitations to keep in mind when interpreting these results. First, the small sample size might impact our results; therefore, a larger sample size should be recruited for future studies. However, our study replicates previous findings in which acute exercise exerts disproportionate benefits on specific types of cognitive function. Furthermore, we administered the cognitive task within 10 min after the exercise session was completed. However, different effects on cognitive functioning might have resulted if measurements were taken during exercise or after a longer delay;[3,4] therefore, the overgeneralization of these results should be avoided. Future research involving various time intervals after acute exercise will be required to examine the durability of this effect. Finally, because few prior studies have examined the relationship between acute exercise and motor response inhibition using electrophysiological approaches, we decided to recruit healthy young adults to establish initial results. However, immature populations (e.g., children) or those with lower or impaired cognitive function (e.g., the elderly) might be influenced differently by acute exercise,[3] so future studies are needed to explore these specific populations.

## 5. Conclusion

Acute, moderately intense exercise may facilitate the motor response inhibition aspect of executive function, as assessed by the stop-signal task. The beneficial effect of acute exercise on inhibition observed in this study corroborates previous findings in which acute exercise facilitated the allocation of attentional resources to inhibition during the go/no-go and Flanker tasks. However, the results of our study further suggest that the effect of acute exercise on inhibition may be task dependent; that is, in the stop-signal task, exercise promotes greater withholding of a response following an inhibitory signal. Lastly, we present a novel finding that the improved motor response inhibition following acute exercise occurs during later cognitive rather than early sensory stages of information processing. Together, our behavioral and electrophysiological results suggest that acute exercise may be useful in facilitating specific aspects of executive function. Further studies will be needed to extend these findings by recruiting a larger sample size from a wider demographic and assessing multiple time points after acute exercise.

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